**6560-50-P**

**Environmental Protection Agency**

**40 CFR Part 63**

**[A-99-03, OAR-2003-0028 FRL-]**

**RIN: 2060-AI72**

**List of Hazardous Air Pollutants, Petition Process, Lesser Quantity Designations, Source Category List**

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Proposed rule.

**SUMMARY**: The EPA is proposing to amend the list of hazardous air pollutants (HAP) contained in section 112(b)(1) of the Clean Air Act (CAA) by removing the compound methyl ethyl ketone (MEK) (2-Butanone) (CAS No. 78-93-3). This action is being taken in response to a petition submitted by the Ketones Panel of the American Chemistry Council (formerly the Chemical Manufacturers Association) on behalf of MEK producers and consumers to delete MEK from the HAP list. Petitions to remove a substance from the HAP list are permitted under section 112(b)(3) of the CAA.

The proposed rule is based on EPA's evaluation of the available information concerning the potential hazards and projected exposures to MEK. We have made an initial determination that there are adequate data on the health and environmental effects of MEK to determine that emissions, ambient concentrations, bioaccumulation, or deposition of the

compound may not reasonably be anticipated to cause adverse human health or environmental effects. This action includes a detailed rationale for delisting MEK, and we request comment on the proposal.

**DATES:** Comments. Written comments on the proposed rule must be received by [INSERT DATE 90 DAYS FROM PUBLICATION OF THIS PROPOSED RULE IN THE FEDERAL REGISTER].

Public Hearing. A public hearing regarding the proposed rule will be held if requests to speak are received by the EPA on or before [INSERT DATE 60 DAYS FROM PUBLICATION OF THIS PROPOSED RULE IN THE FEDERAL REGISTER]. If requested, a public hearing will be held approximately 90 days after the date of publication of this notice in the Federal Register.

**ADDRESSES:** Comments. Comments may be submitted electronically, by mail, or through hand delivery/courier. Electronic comments may be submitted on-line at http://www.epa.gov/edocket/. Written comments sent by U.S. mail should be submitted (in duplicate if possible) to: Air and Radiation Docket and Information Center (Mail Code 6102T), Attention Docket Number A-98-44, Room B108, U.S. EPA, 1301 Constitution Avenue, NW., Washington, DC 20460. Written comments delivered in person or by courier (e.g., FedEx, Airborne, and UPS) should be submitted

(in duplicate if possible) to: Air and Radiation Docket and Information Center (Mail Code 6102T), Attention Docket Number A-98-44, Room B102, U.S. EPA, 1301 Constitution Avenue, NW., Washington, DC 20460. The EPA requests a separate copy also be sent to the contact person listed below (see FOR FURTHER INFORMATION CONTACT).

Public Hearing. If a public hearing is requested by [INSERT DATE 60 DAYS FROM PUBLICATION OF THIS PROPOSED RULE IN THE FEDERAL REGISTER] the public hearing will be held in our EPA Office of Administration Auditorium, Research Triangle Park, NC. Persons interested in presenting oral testimony or inquiring as to whether a hearing is to be held should contact Ms. Kelly A. Rimer, Risk and Exposure Assessment Group, Emission Standards Division (C404-01), U.S. EPA, Research Triangle Park, North Carolina 27711, telephone number (919) 541-2962. Persons interested in attending the public hearing should also contact Ms. Rimer to verify the time, date and location of the hearing.

**FOR FURTHER INFORMATION CONTACT:** Ms. Kelly A. Rimer, Risk and Exposure Assessment Group, Emission Standards Division (C404-01), U.S. EPA, Research Triangle Park, NC 27711, telephone number (919) 541-2962, electronic mail address rimer.kelly@epa.gov.

**SUPPLEMENTARY INFORMATION:**

Regulated Entities. Entities potentially affected by this action are those industrial facilities that manufacture or use MEK. This action proposes to amend the list of HAP contained in section 112(b)(1) of the CAA by removing the compound MEK. The decision to grant the petition and issue a proposed rule to delist MEK removes MEK from regulatory consideration under section 112(d) of the CAA.

Docket. The EPA has established an official public docket for this action under Docket ID No. A-99-03, and Electronic Docket No. OAR-2003-0028. The official public docket is the collection of materials that is available for public viewing at the EPA Docket Center (Air Docket), EPA West, Room B-108, 1301 Constitution Avenue, NW, Washington, DC 20004. The Docket Center is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Reading Room is (202) 566-1744, and the telephone number for the Air Docket is (202) 566-1742.

Electronic Access. An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at http://www.epa.gov/edocket/ to submit or view public comments,

access the index of the contents of the official public docket, and access those documents in the public docket that are available electronically. Once in the system, select "search" and key in the appropriate docket identification number.

Certain types of information will not be placed in the EPA dockets. Information claimed as confidential business information (CBI) and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. The EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the EPA Docket Center.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA

identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

Comments. You may submit comments electronically, by mail, by facsimile, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket identification number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments submitted after the close of the comment period will be marked "late." The EPA is not required to consider these late comments.

Electronically. If you submit an electronic comment as

prescribed below, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. The EPA's policy is that EPA will not edit your comment and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at http://www.epa.gov/edocket, and follow the online instructions for submitting comments. Once in the system, select "search" and key in Docket ID No. A-99-03, or Electronic Docket Id. No.

OAR-2003-0028. The system is an "anonymous access" system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

Comments may be sent by electronic mail (e-mail) to a-and-r-docket@epa.gov, Attention Docket ID No. A-99-03, or Electronic Docket ID. No. OAR-2003-0028. In contrast to EPA's electronic public docket, EPA's e-mail system is not an "anonymous access" system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket and made available in EPA's electronic public docket.

You may submit comments on a disk or CD ROM that you mail to the mailing address identified in this document. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

By Mail. Send your comments (in duplicate, if possible) to: EPA Docket Center (Air Docket), U.S. EPA West, (MD-6102T), Room

B-108, 1200 Pennsylvania Avenue, NW, Washington, DC 20460, Attention Docket ID No. OAR-2003-0028.

By Hand Delivery or Courier. Deliver your comments (in duplicate, if possible) to: EPA Docket Center, Room B-108, U.S. EPA West, 1301 Constitution Avenue, NW, Washington, DC 20004, Attention Docket ID No. OAR-2003-0028. Such deliveries are only accepted during the Docket Center's normal hours of operation.

By Facsimile. Fax your comments to: (202) 566-1741, Docket ID No. OAR-2003-0028.

CBI. Do not submit information that you consider to be CBI through EPA's electronic public docket or by e-mail. Send or deliver information identified as CBI only to the following address: Kelly Rimer, c/o Roberto Morales, OAQPS Document Control Officer (C404-02), U.S. EPA, 109 TW Alexander Drive, Research Triangle Park, NC 27709, Attention Docket ID No. OAR-2003-0028. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

Worldwide Web (WWW). In addition to being available in the docket, an electronic copy of today's proposed rule will also be available on the WWW through the Technology Transfer Network (TTN). Following the Administrator's signature, a copy of the proposed rule will be placed on the TTN's policy and guidance page for newly proposed or promulgated rules at http://www.epa.gov/ttn/oarpg. The TTN provides information and technology exchange in various areas of air pollution control. If more information regarding the TTN is needed, call the TTN HELP line at (919) 541-5384.

Outline. This preamble is organized as follows:

## I. Background

Section 112 of the CAA contains a mandate for EPA to evaluate and control emissions of HAP. Section 112(b)(1) includes a list of 188 specific chemical compounds and classes of compounds that Congress identified as HAP. The EPA must evaluate the emissions of substances on the HAP list to identify source categories for which the Agency must establish emission standards under section 112(d). We are required to periodically review the list of HAP and, where appropriate, revise this list by rule. In addition, under section 112(b)(3), any person may petition us to modify the list by adding or deleting one or more substances. A petitioner seeking to delete a substance must demonstrate that there are adequate data on the health and environmental effects of the substance to determine that

emissions, ambient concentrations, bioaccumulation, or deposition of the substance may not reasonably be anticipated to cause any adverse effects to human health or the environment. A petitioner must provide a detailed evaluation of the available data concerning the substance's potential adverse health and environmental effects and estimate the potential exposures through inhalation or other routes resulting from emissions of the substance.

On November 27, 1996, the American Chemistry Council's Ketones Panel submitted a petition to delete MEK (CAS No. 78-93-3) from the HAP list in section 112(b)(1), 42 U.S.C., 7412(b)(1). Following the receipt of the petition, we conducted a preliminary evaluation to determine whether the petition was complete according to Agency criteria. To be deemed complete, a petition must consider all available health and environmental effects data. A petition must also provide comprehensive emissions data, including peak and annual average emissions for each source or for a representative selection of sources, and must estimate the resulting exposures of people living in the vicinity of the sources. In addition, a petition must address the environmental impacts associated with emissions to the ambient air and impacts associated with the subsequent cross-

media transport of those emissions. We determined the petition to delete MEK to be complete and published a notice of its receipt in the Federal Register on June 23, 1999 at 64 FR 33453 and requested information to assist us in technically reviewing the petition in addition to other comments.

We received ten submissions in response to our request for comment and information which would aid our technical review of the petition. We responded to substantive comments in our technical review of the petition.

## II. Criteria for Delisting

Section 112(b)(2) of the CAA requires us to make periodic revisions to the initial list of HAP set forth in section 112(b)(1) and outlines criteria to be applied in deciding whether to add or delete particular substances. Section 112(b)(2) identifies pollutants that should be listed as:

> . . . pollutants which present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects (including, but not limited to, substances which are known to be, or may reasonably be anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or which are acutely or chronically toxic) or adverse environmental effects whether through ambient concentrations, bioaccumulation, deposition, or otherwise . . ..

Section 112(b)(3) establishes general requirements for

petitioning the Agency to modify the HAP list by adding or deleting a substance. Although the Administrator may add or delete a substance on his or her own initiative, the burden is on a petitioner to include sufficient information to support the requested addition or deletion under the substantive criteria set forth in section 112(b)(3)(B) and (C).

The Administrator must either grant or deny a petition to delist a HAP within 18 months of receipt of a complete petition. If the Administrator decides to deny a petition, the Agency publishes a written explanation of the basis for denial in the Federal Register. A decision to deny a petition is final Agency action subject to review. If the Administrator decides to grant a petition, the Agency publishes a written explanation of the Administrator's decision, along with a proposed rule to add or delete the substance. The proposed rule is open to public comment and public hearing, and all additional substantive information received is considered prior to the issuance of a final rule.

To delete a substance from the HAP list, section 112(b)(3)(C) provides that the Administrator must determine that:

> . . . there is adequate data on the health and

> environmental effects of the substance to determine that emissions, ambient concentrations, bioaccumulation of deposition of the substance may not reasonably be anticipated to cause any adverse effects to the human health or adverse environmental effects.

If the Administrator decides to grant a petition, the Agency publishes a written explanation on the Administrator's decision, along with a proposed rule to add or delete the substance. The proposed rule is open to public comment and public hearing. We evaluate all substantive information received during public comment prior to taking any final action related to a proposed rule.

We do not interpret section 112(b)(3)(C) to require absolute certainty that a pollutant will not cause adverse effects on human health or the environment before it may be deleted from the list. The use of the terms "adequate" and "reasonably" indicate that the Agency must weigh the potential uncertainties and likely significance. Impact of the uncertainties concerning the risks of adverse health or environmental effects may be mitigated if we can determine that projected exposures are sufficiently low to provide reasonable assurance that such adverse effects will not occur. Similarly, impacts of uncertainties due to the magnitude of projected

exposures may be mitigated if we can determine that the levels which might cause adverse health or environmental effects are sufficiently high to provide reasonable assurance that exposures will not reach harmful levels. However, the burden remains on a petitioner to demonstrate that the available data support an affirmative determination that emissions of a substance may not be reasonably anticipated to result in adverse effects on human health or the environment (that is, EPA will not remove a substance from the list of HAP based merely on the inability to conclude that emissions of the substance will cause adverse effect on human health or the environment). As a part of the requisite demonstration, a petitioner must resolve any critical uncertainties associated with missing information. We will not grant a petition to delete a substance if there are major uncertainties that need to be addressed before we would have sufficient information to make the requisite determination.

## III. Summary of the Petition

### A. Background

The petition to delist MEK is presented in the form of a risk assessment that considers multiple routes of exposure and evaluates the likelihood and severity of adverse effects to

human health and the environment arising from exposures to ambient levels of MEK. The petition presents a characterization of the sources and releases of MEK, estimates exposures, identifies the potential hazard and the dose-response relationship of MEK, and characterizes the risk from a reasonable worst-case lifetime exposure to MEK, and to worst-case short-term (24 hour) exposure to MEK. This section of today's proposed action presents an overview of the petition to delist MEK, and the petitioner's conclusions based on that information. Please consult the docket for more detail about the petition or EPA's evaluation of the petition.

The petition to delist MEK presents background information on MEK, including chemical and physical properties data and production and use data. The petitioner used the 1994 Toxic release Inventory (TRI) as the basis of an emissions inventory intended to quantify annual emissions of MEK, to identify and locate emissions sources, and to acquire some facility-specific emissions information. The 1994 TRI shows that there are over 2,000 sources with reported emissions of MEK. The petition states that over 85 percent of these facilities (approximately 1,700) emit 25 tons per year (tpy) or less. The petition also states that approximately 800 facilities emit between 10 and 200

tpy, and 27 facilities emit 200 tpy or more. In addition to using the 1994 TRI, the petitioner queried a subset of individual sources to obtain site-specific source, release, and facility information for the purpose of conducting more detailed risk assessments.

B. Exposure Assessment

The petition's emissions inventory provides the basis for a tiered air dispersion modeling analysis as described in "Tiered Modeling Approach for Assessing Risk due to Sources of Hazardous Air Pollutants" (EPA-450/4-92-001). That tiered analysis applies successive refinements in model selection and input data to derive conservative estimates of the maximum annual average ambient concentration of MEK. "Conservative" refers to the selection of models and modeling parameters that are more likely to overestimate, rather than underestimate, the ambient concentrations of a given pollutant when data are limited.

Tier 1 air dispersion modeling requires limited source information and provides the most conservative estimate of maximum concentrations of the tiers. Tier 2 modeling requires additional source information and a simple air dispersion model and results in air concentrations that are more realistic than

tier 1 estimates, but which are still considered to be conservative. In the assessment, the petition used EPA's SCREEN3 model for tier 2 analyses. Tier 3 requires extensive data from a source and recommends using EPA's most advanced dispersion modeling techniques to provide even more realistic, though generally still conservative, estimates of maximum concentrations. In the assessment, the petitioner used EPA's Industrial Source Complex Short Term 3 (ISCST3) model for the tier 3 analysis. Because each successive tier provides a less conservative and more realistic estimate of the ambient MEK concentration, the petitioner performed tier 3 modeling only where the tier 2 modeling predicted maximum annual average ambient concentrations of MEK above a designated threshold. Using this approach, the petitioner developed a reasonable worst-case exposure scenario by estimating the maximum annual average ambient concentration expected to result from emissions of MEK from a single facility. The petition also accounts for emissions of MEK from several sources located within close proximity to each other (often called a cluster of sources). The petition does this in order to assess the potential impact to a person who may live close to a cluster of MEK-emitting facilities.

The petition reasoned that the majority of risk would come from facilities that emit large amounts of MEK. The petitioner identified facilities which emitted 200 tpy or more of MEK as large. The petitioner contacted each of the 27 large facilities to gather data with which to model maximum, off-site ambient concentrations of MEK. That analysis also used information from title V permits. The petitioner was able to obtain the necessary modeling information for 21 of the 27 facilities, including the six highest emitters of MEK, and 13 of the top 15 emitters. The analysis for these facilities applied tier 2 and tier 3 modeling techniques. The maximum annual average concentration estimated from the largest MEK emission source using the tier 3 model was approximately 1.2 milligrams per cubic meter ($mg/m^3$). However, that concentration was located at the entrance to an adjacent industrial facility where there were no environmental or human receptors. The MEK emissions from the other sources modeled in the tiered approach were all less than 0.9 $mg/m^3$. For the seven facilities with the highest predicted fence line concentrations, the maximum annual average ambient levels of MEK decreased to below 0.5 $mg/m^3$ within 175 meters from the fence line.

In addition to modeling sources emitting 200 tpy or more,

the petition also includes an analysis of sources emitting lesser amounts. The petitioner used a tier 2 analysis to model those MEK sources (approximately 800 in all) which, based on the inventory, emitted more than 10 tpy but less than the 200 tpy. The petitioner divided these emission sources into source categories based on their two digit Standard Industrial Classification (SIC) codes. For each SIC, the petitioner modeled a "worst case" prototype plant using conservative site configurations (e.g., distance to fence lines), the highest reported emissions rate for the individual category, and worst-case dispersion meteorology. The maximum predicted annual average ambient concentration of MEK from the sources emitting less than 200 tpy of MEK was approximately 0.7 mg/m$^3$. The remaining MEK emission sources included under this approach were determined to have maximum annual average ambient concentrations less than 0.6 mg/m$^3$.

The petition includes estimates of 24-hour average concentrations in addition to estimates of annual average concentrations. The highest 24-hour average concentration as predicted by tier 3 modeling was 12.8 mg/m$^3$. That concentration was at the same location where the highest annual concentration was predicted to occur. The petition states that there are no

people or environmental receptors at that location. The petition states further that all other modeled 24-hour concentrations are below 10 mg/m$^3$ and concludes that people would not be exposed to 24-hour concentrations greater than this value.

To address the potential impact of MEK sources that are located within close proximity to each other, the petitioner identified, from the 1994 TRI, every facility in the United States with MEK emissions greater than 10 tpy. The petitioner used postal ZIP codes to determine areas in which emission sources were situated near one another. Using this approach, the petition analyzed 91 facilities. Of these facilities, only three ZIP codes contained groups of facilities that collectively emitted more than 200 tpy. The petitioner used results from the previous tiered analysis to evaluate the potential for these facilities to have significant overlapping impacts. Based on the analysis, the petition concluded that the combined impacts from multiple MEK emission sources situated close to one another will not result in maximum annual average ambient MEK concentrations greater than 1 mg/m$^3$, or in 24-hour concentrations greater than 10 mg/m$^3$. In most cases, the concentrations will be well below these values.

The petitioner reviewed available ambient air monitoring studies to determine the potential contribution of ambient background MEK to the maximum annual average and 24-hour average MEK concentrations. Here, background refers to air concentrations of MEK from sources not modeled in the analysis (e.g., mobile). The review showed that MEK has been monitored in both urban and rural locations. The highest reported MEK concentrations occurred in the Houston ship channel where the yearly averages from 1987-1995 for seven sites ranged from approximately 0.0009 to 0.0018 $mg/m^3$. The maximum 24-hour average concentration also occurred in the Houston ship channel over the same time period where the highest reported average was 0.09 $mg/m^3$. Based on this review, the petitioner concluded that background MEK in not a significant contributor to the maximum annual average, or maximum 24-hour average concentration of MEK.

The petitioner reviewed MEK's fate in the environment to determine the most probable routes of human exposures to ambient MEK. The petitioner used physical chemical data taken from the literature and a number of EPA databases to conclude that MEK does not persist or bioaccumulate in the environment. The petition also states that due to its high vapor pressure, MEK discharged onto a terrestrial environment is expected to rapidly

volatilize to air. Volatilization from water is also reported to occur at a significant rate, and the petition reports MEK to be readily biodegradable in both aerobic and anaerobic environments. The petitioner concluded that MEK is not anticipated to pose an exposure problem in drinking water, and that inhalation is the primary route of exposure for humans living in the vicinity of MEK emission sources.

The petition states that while in the air, MEK decomposes to carbon dioxide, carbon monoxide, and water through various reactions. One of the intermediaries is a probable carcinogen: acetaldehyde. The petitioner maintained that acetaldehyde formed during MEK's transformation disappeared approximately 70 times faster than it was created. Therefore, the petitioner concluded, the rapid dispersion of MEK, coupled with its half-life of about 9 days and the comparatively short half-life of acetaldehyde (about 14 hours), resulted in low ambient levels of MEK-produced acetaldehyde. The petition states that the resulting concentration levels cannot be reasonably anticipated to cause adverse human health effects.

C. Human Health Effects Assessment

The petition presents toxicological data, which are used for hazard identification and to determine dose-response

relationships, citing the EPA's Integrated Risk Information System (IRIS). These data are also supplemented by an extensive review of the literature that includes articles published after the most recent review of the IRIS database for MEK which occurred in 1992.

The petition concludes that MEK's acute and chronic toxicity are low, and that it demonstrates little or no subchronic toxicity. The petition also reports that MEK has been shown to be without genotoxic activity, but it has not been specifically tested for carcinogenicity. However, the petition states that data on MEK's structure, metabolism, subchronic health effects, and genotoxic effects indicate that it is not likely to have carcinogenic properties.

The petition states that MEK by itself has little potential to produce damage to the nervous system. The petition discusses MEK's ability to potentiate the neurotoxic effects of other chemicals when both are present at relatively high concentrations and concluded that MEK does not pose a neurotoxic hazard to humans under ambient exposure scenarios. The petition also states that MEK has not been shown to produce birth defects (i.e., teratogenicity) and does not produce reproductive effects in subchronic inhalation studies.

The petition takes the position that MEK's developmental toxicity is low, and that developmental toxicity is the basis for the 1992 EPA IRIS Reference Concentration (RfC) for MEK of 1.0 mg/$m^3$. The RfC is a peer-reviewed value defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a life time (i.e., 70 years).

The petition provides a review of EPA's derivation of the IRIS RfC for MEK. Based on this review and the application of EPA guidelines that were published after the 1992 update of the MEK RfC, the petitioner proposed a revised criterion for human health effects. The petitioner's proposed revision suggests an increase in the RfC from 1 mg/$m^3$ to 3.3 mg/$m^3$. (The details of the petitioner's reassessment are contained in the docket.)

For short-term exposure, the petition adjusts the revised RfC by eliminating the uncertainty factor of 10 that is used for extrapolating from subchronic to chronic exposure. The resulting short-term human health criterion submitted in the petition is 33 mg/$m^3$.

D. Risk Characterization and Conclusions Regarding Risks to

Human Health

The petitioner characterized human health risks from exposure to the predicted ambient MEK concentration levels by comparing the maximum estimated annual average concentration to their proposed revised RfC of 3.3 mg/m$^3$. Based on the conservatism built into the model estimates, the petitioner concluded that actual maximum annual average ambient concentrations of MEK are unlikely to exceed 1 mg/m$^3$ for the highest emitting source and will be significantly less than 1 mg/m$^3$ for all other sources. The petitioner concluded that the available evidence demonstrates that actual exposures are not likely to approach the 1992 IRIS RfC of 1 mg/m$^3$ and will not exceed the petitioner's revised health criterion of 3.3 mg/m$^3$. The petition characterized human health risks from 24-hour exposures by comparing the estimated 24-hour concentrations, 10 mg/m$^3$ with a human health benchmark of 33 mg/m$^3$, and determined that these short-term concentrations will not approach their criterion of 33 mg/m$^3$. Therefore, the petitioner concluded that adverse human health effects arising from ambient exposures to MEK emissions cannot be reasonably anticipated to occur.

E. Ecological Assessment and Conclusions

The petition presents ecological toxicity data for

environmental effects as the basis for its assessment of the potential ecological risks from the release of MEK to the environment. The petition uses data from several EPA databases and from the general literature. The petition includes no data on the potential for ecological effects to occur due to its presence in media other than water. The petitioner concluded that the available data indicate that MEK has low acute toxicity for aquatic organisms. Although there are no data on chronic aquatic toxicity, the petitioner stated that MEK is not expected to be chronically toxic to aquatic organisms because of its limited persistence in aqueous habitats, which results from its rapid volatilization and biodegradation. The petition compares predicted maximum ambient annual average concentrations to the identified ecotoxicity endpoints. Based on that comparison and information on MEK's environmental behavior, the petitioner concluded that MEK cannot reasonably be anticipated to cause significant and widespread adverse environmental effects.

## IV. EPA Analysis of the Petition

The following section presents EPA's evaluation and analysis of the petition to delist MEK. The technical review was conducted by EPA's Office of Air and Radiation, with assistance from EPA's Office of Research and Development. The

supporting review materials are contained in the docket.

A. Exposure Assessment

Methyl Ethyl Ketone is a clear, colorless, stable, low-boiling point (79.6 ºC), highly volatile (vapor pressure 90.6 torr at 25 ºC), highly flammable (flash point 1 ºC, auto ignition temperature 515 ºC) liquid. It is very soluble in water (240 grams per liter at 20 ºC), miscible with organic solvents and forms azetropes with water and many organic liquids. Methyl ethyl ketone has exceptionally high solvent powers for many natural and synthetic resins. It is used as a solvent in the surface coatings industry, specifically in vinyl lacquers, nitrocellulose lacquers, and acrylics, and is used as a chemical intermediate. Methyl ethyl ketone is also used in other industries for producing adhesives, magnetic tapes, printing inks, degreasing and cleaning fluids, as a dewaxing agent for lubricating oils, as an intermediate in the production of antioxidants, perfumes, and as a catalyst. Methyl ethyl ketone also occurs naturally. It is emitted from various evergreen trees and has been identified as a natural component of several foods.

We concur with the petition that inhalation is the principal route of non-occupational exposures to MEK emissions.

The absorption of MEK through the skin at the estimated ambient levels is likely to be insignificant compared to inhalation. In addition, its relatively rapid volatilization and rapid biodegradation in water indicates that humans are unlikely to be exposed to significant amounts of MEK in drinking water.

To determine the adequacy of the petition's exposure assessment, we first evaluated the emissions inventory and the petition's source characterization. We then evaluated the dispersion modeling in terms of the methods and application of the models.

To evaluate the emissions inventory, we compared the petition's list of MEK emission sources to EPA's 1996 National Toxics Inventory (NTI). We determined that the petition correctly identified the largest sources of MEK emissions, and that the quantity of emissions for each identified source was comparable to the NTI. There was an overall lack of agreement, however, between the total count of MEK emission sources listed in the NTI and in the petition's inventory. We determined that this resulted from a general weakness in the ability of the petitioner's approach to identify facilities emitting less than 25 tpy of MEK. However, after reviewing both the inventory and the petitioner's tiered modeling approach, we determined that

these discrepancies are not material to the subsequent exposure analysis, and agreed that we would consider the characterization of the maximum concentrations from the medium and large sources to account for the reasonable worst-case exposure scenario. Therefore, we have concluded that the petitioner's emissions inventory provides an adequate basis for the dispersion modeling and exposure assessment presented in the petition.

To evaluate the petition's characterization of sources (e.g., stack heights, plume rise, distance to the nearest fence line and meteorology), we considered the petitioner's use of the TRI database and acquired a subset of the parameters the petitioner used in the more site-specific (tier 3) assessments. We determined that the petitioner appropriately used TRI as a basis for characterizing sources. We examined the source parameters the petitioner used in the tier 3 analyses and determined, based on our engineering knowledge of the types of sources included in the analyses, that the parameters are reasonable.

Our evaluation of the petition's dispersion modeling approach initially focused on the petitioner's use of the EPA models in the tiered analyses. We evaluated the petition's modeling approach for both annual average concentrations and for

24-hour concentrations. Our evaluation verified that the petitioner applied appropriate EPA guidelines in the modeling effort, and that the data inputs used in the models are appropriately conservative.

We first evaluated the petition's modeling of long-term averages. To develop a more detailed evaluation of the petition's dispersion analyses, we acquired from the petitioner electronic copies of the raw data inputs and the model runs for seven of the largest emissions sources. This represents a subset of the sources which emit over 200 tpy. The EPA selected these sources for scrutiny from the tier 3 analysis set which the petitioner modeled using EPA's ISCST3 model. Based on a detailed review of the data inputs and the ISCST3 model runs, we confirmed that a conservative estimate (i.e., more likely to be over predicted than under predicted) of the highest maximum annual average concentration of MEK for all the facilities modeled is approximately 1.2 $mg/m^3$. We agree with the petitioner's assertion that this concentration occurred at the entrance to an industrial facility adjacent to a relatively large MEK emission source in an industrial park. The maximum annual average concentration for the remaining emissions sources were all less than 0.9 $mg/m^3$.

We confirmed that for this subset of emission sources, the maximum predicted annual concentration of MEK declined below 0.5 $mg/m^3$ within 175 meters of the facility fence lines. Therefore, we concur with the petitioner that the predicted concentrations decline rapidly as the distance from the emission source increases. That is, within the relatively short distance of 175 meters, the maximum annual concentrations of MEK are likely to be at least a factor of two lower than the maximum predicted ISCST3 values for all sources in this subset.

We evaluated the petitioner's modeling analyses for sources emitting less than 200 tpy of MEK. The petitioner used a tier 2 analysis to predict maximum annual average concentrations for a series of worst-case emission scenarios for this subset of sources. After a detailed evaluation of the model parameters and input data, we determined that the petitioner's analyses of these emission sources also followed the appropriate EPA dispersion model guidelines.

Based on our review, we have concluded that the predicted maximum annual average concentration for those sources emitting less than 200 tpy of MEK is less than 0.7 $mg/m^3$. These predicted concentration levels are conservative estimates which are also expected to decline rapidly as distance from the facility

increases.

During the review, we questioned the petitioner's designation of "large emission sources" as those sources emitting more than 200 tpy of MEK. We requested that they conduct a more detailed analysis on sources emitting less than 200 tpy. We suggested that the petitioner use a minimum emission rate that could theoretically result in an exceedance of the petition's own specified health criterion of 3.3 mg/m$^3$. The petitioner would then assess the impact of this new "threshold of significance" on the number and identity of sources in the "large emission sources" category and, if appropriate, reassess the impacts of this change on concentrations of ambient MEK.

To accomplish this, the petitioner used very conservative assumptions of stack height, plume rise, meteorology, and distance to fence line to define a worst-case facility. Using this worst-case emission scenario coupled with EPA's SCREEN3 model, the petitioner demonstrated that sources emitting less than 90 tpy could not reasonably be expected to exceed the petition's proposed criterion of 3.3 mg/m$^3$. The petitioner then updated the emissions inventory using the 1996 TRI to identify those sources emitting between 90 and 200 tpy of MEK.

The petitioner then revised the “threshold of significance” to reflect the use of the 1992 IRIS RfC of 1 mg/m$^3$ as a decision criterion. To derive the new threshold, the petitioner decreased some of the conservatism in the tier 1 parameters and remodeled a new worst-case scenario. The petitioner determined that with this new set of assumptions, emissions greater than 145 tpy would be necessary to exceed a 1 mg/m$^3$ criterion. However, rather than restrict the new analysis to only those sources emitting between 145 and 200 tpy, the petitioner chose to evaluate the larger range of emission sources. Consequently, the revised dispersion modeling analysis focused on those sources emitting between 90 and 200 tpy of MEK. The petitioner submitted that analysis to EPA as an addendum to the original petition.

The petitioner’s approach in the revised modeling analysis was to limit the potential for the model to overestimate exposure (compared to the original modeling approach), while maintaining adequate levels of conservatism in the final estimate. To accomplish that, the petitioner quantified the degree of overestimation in the previous modeling approaches due to conservative source-receptor configurations and adjusted to current model accordingly. That adjustment removed one level of

conservatism from the estimates and provided a more realistic, but still conservative, estimate of the maximum annual average concentrations. The adjustment was applied to each of the emission sources in the previous analysis for those sources emitting from 90 to 200 tpy.

Based on this approach, the petitioner estimated that the maximum annual average concentration for the 18 facilities identified which emitted between 90 and 200 tpy of MEK would be less than 0.96 mg/$m^3$. This value occurred at only one emission source; the remaining 17 facilities in the 90 to 200 tpy range were all less than 0.75 mg/$m^3$.

We conducted a detailed review of the revised analytical approach and determined that it was acceptable. To quantify the conservatism of the adjusted model outputs, we recommended a site-specific analysis using an ISCST3 model (i.e., tier 3) of the source with the highest estimated MEK concentration (i.e., 0.96 mg/$m^3$) after the adjustment. The tier 3 analysis predicted a maximum annual average concentration of 0.17 mg/$m^3$ of MEK from that facility. The tier 3 estimate was then compared to the adjusted emissions estimates to determine the extent of the conservatism remaining in the adjusted estimates. That comparison indicated that the petitioner’s adjusted approach

overestimated maximum annual average concentration for the source by approximately a factor of six.

The petitioner provided the tier 3 analysis and the supporting data for our evaluation. After reviewing the model run and the supporting documentation in detail, we concluded that the petitioner's approach applies appropriate EPA guidelines and adequately characterizes maximum MEK concentrations from industrial sources. Therefore, based on that information, we have concluded that the maximum annual average MEK concentration from facilities emitting between 90 and 200 tpy of MEK may not reasonably be anticipated to exceed 0.96 $mg/m^3$, and we expect it to be much less in most cases.

We used the petition's information on the identity and location of MEK facilities to assess the impacts of sources located in close proximity to one another. Using a tier 2 analysis, we independently modeled the emissions from nine sources located relatively close to one another in two adjacent postal ZIP codes. Our analysis confirmed that MEK disperses rapidly as the distance from the emission source increases, and that at the point of maximum impact, the maximum annual average MEK concentration from multiple sources located close to each other may not reasonably be anticipated to exceed 1 $mg/m^3$; in

fact, we expect it to be much less than 1 mg/m$^3$.

To evaluate the potential contribution of the ambient background MEK to the maximum annual concentration of MEK, we reviewed the literature and various databases, including our Aerometric Information Retrieval System (AIRS) monitoring database and the California Air Toxics database. The available data show MEK measurements ranging from nondetectable to a high of 0.002 mg/m$^3$ reported in AIRS. That value occurred in the Houston ship channel and represents mean concentrations, averaged over 1 year, from seven sites for the years 1987-1995. In addition, the 2001 AIRS entries show similar maxima (e.g., AIRS shows averages 0f 0.002 mg/m$^3$ from sites in Providence, Rhode Island). Based on that review, we have concluded that background concentrations are not likely to have a significant influence on maximum annual exposures to MEK.

Given that the petitioner used the same modeling approach to predict 24-hour concentrations as was used to predict annual average concentrations, we accept the conclusion that the maximum 24-hour average concentration expected would be less than 10 mg/m$^3$. However, we also wanted to evaluate predicted concentrations which may occur over a 1-hour time period. Using air dispersion modeling principles described in EPA's SCREEN3

User's Manual and the estimated annual average and 24-hour average concentrations presented in the petition, we estimated the maximum 1-hour concentration. The predicted annual average concentration is approximately 1 mg/m$^3$ and the 24-hour average is about 10 mg/m$^3$. To estimate the 1-hour maximum, we multiply the 24-hour average by 2.5. This results in a 1-hour maximum of approximately 25 mg/m$^3$.

In terms of ambient air monitoring data, the 2001 AIRS shows that the highest 24-hour concentration is 0.03 mg/m$^3$, and the highest 3-hour concentration is 0.06 mg/m$^3$. Both of these concentrations were monitored in Rhode Island at the same location as the highest annual average concentrations for the year 2001. As with the annual average monitoring data, these short-term values are sufficiently low so as not to contribute significantly to short-term maximum concentrations.

To summarize, the petitioner developed a tiered modeling analysis of MEK emissions using EPA's tiered approach to regulatory models. We determined that the petitioner performed all analyses following EPA modeling guidelines, and that the results provide conservative estimates of ambient levels of MEK from the inventoried sources. The modeling study demonstrated that, with the exception of the one location (at the entrance to

a facility in an industrial park), estimated maximum annual average concentrations of MEK were less than 1 mg/m$^3$ for all facilities modeled, and well below 1 mg/m$^3$ for most of the facilities modeled. For 24-hour and 1-hour averages, we expect the concentrations would not exceed 10 and 25 mg/m$^3$, respectively. Also, based on the location of the maximum annual and 24-hour off-site concentration predicted at the highest emitting facility, EPA has concluded that no individual could be reasonably anticipated to experience chronic or 24-hour exposures at the level of the predicted maximum ambient concentrations. Therefore, given the conservatism built into the models and petitioner's modeling assumptions, EPA has concluded that we may not reasonably anticipate maximum annual exposures to MEK to exceed 1 mg/m$^3$. In addition, based on the evaluation of multiple sources located relatively close together, we may not reasonably anticipate that the collective emissions of MEK will result in a maximum annual average off-site concentration of MEK greater than 1 mg/m$^3$, or a 24-hour average greater than 10 mg/m$^3$. We, by extrapolation, have concluded that 1-hour concentrations from multiple sources would not exceed 25 mg/m$^3$. Finally, the petitioner's use of air concentrations for each emission source to characterize the

exposed population is an acceptable, conservative approach to exposure modeling. That is, an exposure assessment that would estimate exposures for actual people living near these emission sources would likely result in maximum individual exposures from ambient air that are lower than the estimates presented in the petition. Given the likely proximity of inhabitable areas and the variability of human activity patterns, it is our expectation that actual maximum individual exposures would be up to a factor of ten less than the maximum exposures presented in the petition. Therefore, in light of our review of the petitioner's exposure analysis, we have concluded that exposures to annual average ambient concentrations of MEK may not reasonably be anticipated to exceed 1 $mg/m^3$, and that the maximum 24-hour exposures may not reasonably be anticipated to exceed 10 $mg/m^3$. Also, based on our own analysis, we have concluded that maximum 1-hour exposures may not reasonably be anticipated to exceed 25 $mg/m^3$.

B. Human Health Effects Assessment

We determined that the petition uses the same toxicological database as the 1992 IRIS assessment of MEK to characterize human health effects and to identify an appropriate human health criterion for the risk characterization for chronic

effects. The IRIS is the Agency's official repository of consensus human health risk information. It was created and is maintained by the Agency to provide assistance to Agency decision makers on the potential adverse human health effects of particular substances. In addition, we evaluated recent studies reported in the published literature.

Methyl ethyl ketone is classified in the IRIS (1992) as a Group D compound. A Group D compound is one that is not classifiable as to human carcinogenicity. This classification is based on the absence of human carcinogenicity data and inadequate animal data. There are no animal cancer bioassays of MEK by either the oral or inhalation route. There are structural data on MEK. One study concludes that MEK is unlikely to be carcinogenic based on the lack of any structural features or alerts indicative of carcinogenic potential as a result of mechanism-based structure-activity relationship (SAR) analysis (Woo et al., 2002). Further, Woo has given MEK a low concern rating (unlikely to be of cancer concern) based on comparison to acetone for which there is no evidence of carcinogenicity, and the fact that there is no evidence that unsubstituted mono-ketones have been associated with carcinogenicity/genotoxicity. There is also no reason to

anticipate any electrophillic reactivity for unsubstituted mono-ketones mentioned above (i.e., no structural alerts).

Cancer data on humans from which to draw conclusions about potential carcinogenic risks to the human population are weak and limited. None of the occupational epidemiology studies we examined (four studies of three different worker cohorts were available) provided clear evidence of increased cancer risk from occupational exposure to MEK. These data do provide some suggestion of evidence of an increased risk between multiple solvent exposures which included MEK and some cancers including bone and prostate cancer. (Alderson and Rattan, 1980; Wen et al., 1985; Spirtas et al., 1991; Blair et al. 1998.)

One study that has received some attention is a 1987 study investigating potential carcinogenic effects in the children of males occupationally exposed to MEK (Lowengart et al., 1987). This study included 123 matched pairs of children whose fathers reported, by questionnaire only, occupational exposure to various compounds including MEK, chlorinated solvents, spray paints, dyes and pigments, and cutting oils. The study reported a statistically significant positive trend for risk of childhood leukemia based on father’s frequency of use for all of the chemicals mentioned, including MEK. Paternal exposure to MEK

also appeared elevated, but not statistically significantly so, for the period of paternal exposure after birth of the child but not during pregnancy or one year before pregnancy. This study is considered as an exploratory study, based solely on questionnaires with no other exposure information. Factors that could be confounding covariates such as exposures to other chemicals and personal lifestyle were not taken into account in the statistical analysis of this study.

Methyl ethyl ketone has been tested for activity in an extensive spectrum of in vitro and in vivo genotoxicity assays and has shown no evidence of genotoxicity in most conventional assays (National Toxicology Program, no date; World Health Organization 1992; Zeiger et al., 1992). Methyl ethyl ketone tested negative in bacterial assays (both the S. typhimurium (Ames) assay, with and without metabolic activation, and E. coli), the unscheduled deoxyribonucleic acid (DNA) synthesis assay, the assay for sister chromatid exchange (SCE) in Chinese hamster ovary (CHO)cells, the mouse lymphoma assay, the assay for chromosome aberrations in CHO cells, and the micronucleus assay in the mouse and hamster. The only evidence of mutagenicity was mitotic chromosome loss at high concentrations in a study of aneuploidy in yeast S. cerevisiae; the relevance

of this finding to humans is questionable. Overall, studies of MEK yield little or no evidence of genotoxicity.

Overall, the epidemiologic evidence is weak from which to draw conclusions about the carcinogenic risk in the human population. While none of the studies provides clear evidence of an increased cancer risk, with the totality of the evidence considered inconclusive, the data do provide some suggestion of an increased risk between multiple solvent exposures which include MEK and cancer, specifically childhood leukemia, bone cancer and prostate cancer. There is, however, an absence of positive results in the majority of mutagenicity and genotoxicity tests which are designed to indicate the potential for carcinogenicity, and there is a lack of structural features or alerts indicative of carcinogenic potential in SAR analysis. Based on these results we believe that MEK may not reasonably be anticipated to be carcinogenic.

Developmental toxicity was the basis for the IRIS RfC of 1 $mg/m^3$ which was verified in 1992. The critical study in the derivation of the RfC involved Swiss mice that were exposed to 0; 1,174; 2,978; or 8,906 $mg/m^3$ MEK for 7 hours per day during gestation days 6 through 15 (Schwetz et al., 1991). Neither material nor developmental toxicity was observed at the low- or

mid-doses. At the highest dose, there was a decrease in fetal body weight that was significant only in males. There was also a significant trend in the incidence of misaligned sternebrae when measured on a fetus but not a litter basis. At the highest dose, there was also an increase in relative liver and kidney weight, but the toxicological significance of that effect, if any, is reported in the IRIS as unknown. The lowest observed adverse effect level (LOAEL) for this study was 8,906 mg/m$^3$, and the no observed adverse effect level (NOAEL) was 2,978 mg/m$^3$.

The available data indicate that MEK is not likely to be a reproductive toxicant. There exists no inhalation reproductive toxicity study of MEK; however, an oral two-generation reproductive/developmental toxicity study of 2-butanol, a metabolic precursor to MEK, is available and is the basis for the oral reference dose (RfD) for MEK (Cox et al., 1975). 2-Butanol is quantitatively converted to MEK within the body. In this two-generation study, administration of 2-butanol to rats in drinking water at concentrations as high as 3 percent (~5000 mg kilograms-day) did not affect reproductive performance, but did induce developmental effects consistent with the results from inhalation developmental toxicity studies in rodents. The absence of any pathological lesions in the reproductive organs

of rats exposed to MEK by inhalation for 90 days to concentrations as high as 14,865 mg/m$^3$ also provides some indication that MEK is not likely to be a reproductive toxicant.

The IRIS assessment of MEK states that at present, there is no convincing experimental evidence that MEK is neurotoxic ". . . other than possibly inducing CNS (central nervous system) depression at high exposure levels." The IRIS documentation shows that no peripheral neurohistopathological changes were reported in rats exposed continuously to 3,320 mg/m$^3$ MEK for up to 5 months (Saida et al., 1976). No treatment-related central or peripheral neurohistopathology was observed in rats exposed for 90 days (6 hours/day, 5 days/week) at concentrations of MEK as high as 14,865 mg/m$^3$, even among animals specifically prepared and examined for neurohistopathology (Cavender et al., 1983). Also, ten of ten rats exposed to MEK at 17,700 mg/m$^3$ and higher for 8 hours/day, 7 days/week, died in the 7$^{th}$ week of exposure without neurological symptoms or histopathology (Altenkirch et al., 1978).

Methyl ethyl ketone has been shown to potentiate neurotoxicity of other solvents in experiments with laboratory animals when both MEK and the other solvent are present in high concentrations. The EPA addressed the issue of interactions

such as this in the text of the prospective RfC. We described several studies with human volunteers (see Dick et al., 1992, and references therein) that have MEK exposure groups (at 100 parts per million (ppm) coexposed to relatively low levels, also around 100 ppm) of several other solvents including acetone, methyl isobutyl ketone and toluene. At least for the brief exposure periods in those studies (around 4 hrs), the authors observed no evidence of neurotoxic interactions. However, a recent review (Noraberg and Arlien-Soborg, 2000) reports evidence of possible interactions even at occupational concentrations below the threshold limit values (TLV) (200 ppm, 590 mg/$m^3$) in solvent mixtures containing MEK at 200-300 ppm and n-hexane at 60 ppm. This point should be considered when evaluating mixtures of solvents, especially those containing MEK and the solvents listed above, especially n-hexane. However, the lower limits of MEK exposure that may result in potentiation with other solvents have not been well established, and the potential of MEK in this regard remains a concern, although a minor one. Such concerns are especially diminished at the low-levels we are concerned with in this assessment (i.e., much less than 590 mg/$m^3$).

The petition presents a short-term criterion of 33 mg/$m^3$,

which is an adjustment of their RfC of 3.3 mg/m$^3$. The petitioner calculated this value by simply eliminating the uncertainty factor of ten that is used for extrapolating from subchronic to chronic exposure in the RfC. We do not agree that this is an appropriate method of arriving at an short-term human health effects criterion, however, currently there is no EPA human health criterion for short-term exposures available for us to use in an analysis.

There are 1999 California Environmental Protection Agency (CalEPA) short-term health criteria (CalEPA 1999). The CalEPA published three levels of acute reference exposure levels (REL) to protect against mild adverse effects (associated with a 1-hour exposure), severe effects (associated with a 7-hour exposure), and life threatening effects (associated with a 1-hour exposure). The REL for mild effects is 13 mg/m$^3$, for severe effects it is 32 mg/m$^3$, and for life threatening effects it is 1,385 mg/m$^3$. For the purposes of our analysis and decision, we focused on the mild REL, to be health protective. The CalEPA acute REL to protect against mild effects is based on the study of Nakaaki (1974). However, we consider the results with MEK from the studies of Dick et al. (1984, 1988, 1989, 1992) to be more scientifically defensible for the purposes of

our analysis. Compared to the Nakaaki study, the Dick et al. studies tested more subjects (20+ per study versus four), used control groups extensively, better controlled the exposures (constant in the Dick et al. studies versus increasing concentrations in Nakaaki), analyzed a greater number of endpoints, and apparently longer duration exposures. Collectively, the volunteer studies of Dick et al. indicate that exposures to MEK of up to 200 ppm (590 mg/m$^3$) and up to 4 hours would be an acceptable nonadverse effect concentration in the general population for both subjective effects (such as objectionable odor or irritancy) and for neurobehavioral effects. We would expect the same nonadverse effect concentrations to be relevant for children, as there is no reason to consider children as a sensitive subgroup for such a highly subjective, nonadverse effect as mild irritancy.

C. Determination of an Appropriate Health Effects Criterion for Chronic Noncancer Effects

For risk assessments which estimate chronic noncancer effects from inhalation exposures, the IRIS inhalation RfC is the primary quantitative consensus value used by the Agency.

The RfC for MEK of 1 mg/m$^3$ was placed on IRIS in 1992. It was derived from the Schwetz et al. (1991) developmental

toxicology study by dividing the NOAEL (2,978 mg/m$^3$) by a series of uncertainty factors (UF). The UF for the determination of the MEK RfC was 3,000. This overall uncertainty factor reflects uncertainties in interspecies extrapolation (UF=10), sensitive individuals (UF=10), and an incomplete database, including a lack of chronic and reproductive toxicity studies (UF=10). In addition, a modifying factor (MF=3) was used to account for the absence of unequivocal data for portal-of-entry effects. This resulted in a combined UF and MF of 3,000.

It is Agency policy that the IRIS represents a starting point for risk assessments, however, it is not given conclusive weight in the context of rulemaking. If an outside party questions information presented in the IRIS, we will consider all credible and relevant information before us in the course of making our decision.

Accordingly, the petitioner reviewed the IRIS RfC in light of guidelines published by EPA in 1994, which addressed and updated methods for calculating RfC. Applying these guidelines to the same critical IRIS developmental study used to derive the IRIS RfC, which used the older methodology, the petitioner proposed a revised health criterion based on a reduction of the MEK uncertainty factor for interspecies extrapolation. This

involved a reduction of the interspecies UF of 10 to a default value of 3. The reduction in the interspecies UF is consistent with the guidelines and is warranted if standard default dosimetric adjustments are incorporated in the original study. As a result, the petitioner proposed a revised RfC value of 3.3 $mg/m^3$ (which we view as being equivalent to 3 $mg/m^3$ since EPA generally expresses the RfC as a whole number).

The EPA's Office of Research and Development (ORD) reviewed the petitioner's proposed revision to determine whether such an alternative RfC was appropriate. That review indicated that the method that the petitioner applied to derive the criterion was consistent with both EPA policy and guidance. However, ordinarily, it is Agency policy that revisions in the IRIS are performed such that the entire database is simultaneously reevaluated for all effects and for all routes of exposure. This is done for both administrative efficiency and to ensure that we evaluate the breadth of available science.

Subsequently, EPA announced in the Federal Register (67 FR 1212, January 9, 2002) that it would undertake a formal IRIS review of MEK. The announcement recognized that in the decade since the initial IRIS assessment of MEK, substantive alterations in the Agency's methods for dose-response

assessments have occurred. The estimated completion date for the assessment, including peer review and external peer review is September 2003. We will consider the results of that review prior to taking any final action related to the proposed rule.

In the meanwhile, to support statutory requirements and assist in the determination of the technical merits of the petition to delist MEK, EPA's ORD initiated a parallel undertaking to derive an interim health effects threshold for MEK inhalation exposure that incorporates consideration of current data and current EPA science policy. This process has resulted in the derivation of a prospective RfC of 9 mg/m$^3$. The analysis underlying the development of this prospective RfC can be found in "A Prospective Reference Concentration for MEK (78-93-3)" which is in the docket for today's proposed action.

We consider this prospective RfC to be the most complete and current dose-response information on MEK and, therefore, have determined that it is the appropriate chronic noncancer health effects criterion for EPA to use in today's proposal to remove MEK from the HAP list. In our final evaluation about the potential for MEK to cause noncancer health effects, we will rely on the final RfC and other information resulting from the completed IRIS assessment. Thus, we will not take final action

on today's proposed rule until such information becomes available. In today's action, we request comment generally on our prospective RfC and on the portion of our human health risk characterization based on this RfC. Also, because we recognize that there is some possibility that the RfC may change, we solicit comment on whether it would be appropriate for the Agency to delist MEK if the final RfC is different from the prospective RfC; for example, if it is finalized at 3 mg/m$^3$, the level suggested by industry in its petition, or if it remains unchanged from the 1992 RfC of 1 mg/m$^3$.

The prospective RfC is based on the same critical study as the 1992 IRIS. Consistent with recent Agency recommendations for developing RfD and RfC, the assessment incorporates a duration adjustment to the critical study's NOAEL. This approach adjusted the discontinuous inhalation exposure (7 hours per day) in the critical study to a continuous (24 hours per day) duration. This procedure is premised on a simple concentration x time relationship, and it had the effect of reducing the adjusted NOAEL to 863 mg/m$^3$ from the value of 2,978 mg/m$^3$ used in developing the 1992 RfC.

Using the adjusted NOAEL, the assessment derives a human equivalent concentration (HEC) for MEK. The HEC represents an

external air concentration estimated to achieve the same blood levels in humans and animals. Based on the available blood-to-air coefficient data for MEK in animals and humans, EPA applied the default factor of one for this derivation which resulted in a $NOAEL_{HEC}$ of 863 mg/$m^3$. As with the standard IRIS assessment, EPA applied uncertainty factors to the $NOAEL_{HEC}$ to account for recognized areas of uncertainty in extrapolating the data to the appropriate human scenario. The EPA concluded that the 1992 IRIS interspecies uncertainty (UF=10) and the modifying factor (MF=3) should be revised. However, we concluded that the intraspecies uncertainty (UF=10) should remain unchanged.

The EPA applied the Agency's 1994 RfC methodology to the prospective RfC which results in an interspecies uncertainty factor of three. The prospective RfC also eliminates the previous modifying factor (MF=3) included in the 1992 IRIS to account for the absence of unequivocal data for portal-of-entry (respiratory tract) effects. This revision was, in part, due to additional information in a 1992 National Institute for Occupational Safety and Health (NIOSH) study in which 24 volunteers exposed to 590 mg/$m^3$ of MEK for 4 hours reported no net complaints of even minor irritation. The consequence of that study was a decrease in the uncertainty around irritant

type of portal-of-entry effects in humans.

The prospective RfC also addresses the 1992 IRIS database uncertainty factor (UF=10). The assessment states that the problematic situation that existed in 1992 persists; namely, the difficulty of establishing a health-based guideline for a lifetime chronic exposure without any toxicity studies involving lifetime chronic exposures. The existing long-term repeated exposure experiments have certain flaws that affect their use in developing an inhalation RfC. However, the assessment concludes that EPA can use information from existing studies, as well as ancillary information from new sources, to reduce the concerns in the database. The assessment concludes that the analysis, coupled with the totality of the other available information, has the overall effect of reducing uncertainty in the database such that it is appropriate to apply a partial database uncertainty factor of three, rather than a full database uncertainty factor of ten, in developing the prospective RfC.

This reduction, taken with the reduction in interspecies UF and the elimination of the modifying factor, reduced the composite uncertainty from 3,000 to 100. Therefore, EPA concludes that the prospective RfC is 9 $mg/m^3$.

D. Human Health Risk Characterization and Conclusions

Methyl ethyl ketone is currently listed in IRIS based on a 1989 evaluation as “not classifiable as to human carcinogenicity” according to the 1986 Cancer Guidelines. The IRIS summary identified the lack of both animal and human data to assess the carcinogenic potential of MEK, and at the current time, animal cancer bioassays with MEK by either the oral or inhalation route are still lacking, and there are no indications that such studies are either ongoing or planned. However, genotoxicity information does not indicate any readily apparent genetic mechanism of action for MEK, and the existing genotoxicity tests for MEK are essentially negative. In addition, structural data on MEK do not support any readily apparent basis for a carcinogenic hazard.

The retrospective cohort studies of worker populations exposed to MEK provide no clear evidence of a cancer hazard in these populations. Because of various study limitations, these studies are weak and cannot support conclusions about the carcinogenic potential of MEK in humans. A case-control study examining the association between paternal exposures to several solvents including MEK and childhood leukemia is exploratory in scope such that we cannot use the

results to reliably support the existence of any such association. Overall, this epidemiologic evidence is inconclusive and weak from which to draw conclusions about carcinogenic risks in the human population, although there is some suggestion between increased risk for some cancers and multiple solvent exposures, which included MEK. However, we consider the inconclusive nature of these studies to be offset by more conclusive results regarding the low potential of MEK to be carcinogenic, including the overall lack of positive results from genotoxicity tests and mutagenicity tests, and the lack of any indication of carcinogenicity from structure-activity relationships. Consequently, we conclude that we may not reasonably expect MEK to be carcinogenic.

In the analysis, we use a hazard quotient (HQ) approach to characterize the chronic noncancer risk associated with the exposure to MEK. The HQ is the ratio of a level of exposure for a given substance over a specific time period to a health criterion or reference level for that substance derived from a similar exposure period. We use the maximum annual average ambient concentration as the exposure for the purposes of the chronic HQ calculation. We use EPA’s prospective RfC as the chronic health criterion, and we also calculate an HQ using the

petitioner's RfC. These criteria encompass a 70-year lifetime of continuous exposure and address the health effect of concern due to chronic inhalation exposures to MEK. In addition, the criteria include the margins of safety built into the IRIS RfC and are, therefore, protective of sensitive subpopulations.

Based on our evaluation of the modeling data presented in the petition, we judge that maximum ambient annual exposures from stationary sources to MEK are not likely to exceed 1 $mg/m^3$. Using EPA's prospective RfC of 9 $mg/m^3$, the HQ for the maximum annual average ambient exposure to MEK is 0.1. This means that a person's maximum exposure would be 10 percent of the RfC.

We judge that the exposures to MEK of actual persons living in the immediate vicinity of an MEK emission source would more typically be at least a factor of two to ten less than 1 $mg/m^3$. Therefore, replacing the maximum ambient concentration with a more realistic exposure scenario yields an HQ less than 0.1. Based on the current information, and given the conservative nature of the parameters used to estimate the maximum exposure, the protective nature of the prospective RfC, and because the petition and subsequent analyses characterize the vast majority of MEK exposures from stationary sources, we conclude that by applying the prospective RfC of 9 $mg/m^3$,

potential ambient exposures to MEK may not reasonably be anticipated to cause adverse human health effects.

With regard to the potential for short-term exposures to MEK to result in adverse health effects, we draw a qualitative conclusion. From the petition's modeled 24-hour maximum concentration of 10 mg/m$^3$, and using the conversion factor from EPA's SCREEN3 model User's Guide, we estimate that the maximum 1-hour concentration would not exceed 25 mg/m$^3$. From the Dick et al. study, we see that exposures to MEK of up to 590 mg/m$^3$ and up to 4 hours did not cause adverse effects to human subjects. While we have not developed a short-term human heath criterion from that study, we consider the gap between the adverse effects level in the Dick et al. study and the 24-hour and 1-hour concentrations to be large enough that we may not reasonably anticipate adverse effects to occur from these exposures. Further, as we state above, we consider the maximum annual average concentration estimates to be overestimates of true exposure. Given that the 24-hour and 1-hour ambient air concentrations were estimated using the same information and methods as the annual average concentrations, we consider these short-term concentrations to be similarly conservative. This provides us with additional confidence that adverse effects from

short-term exposures will not occur.

As discussed previously, we will consider the final RfC that results from the IRIS review and substantive public comment as that information becomes available. In addition, we expect to receive information on MEK from industry's submittal to the Agency's Voluntary Children's Chemical Evaluation Program (VCCEP). The VCCEP is intended to provide information to enable the public to understand the potential health risks to children associated with exposures to certain chemicals. Under that program, EPA has asked industries which manufacture or import certain chemicals to develop assessments regarding the potential health effect, exposures, and risks of those chemicals to children. We anticipate industry's submission to the first tier of the VCCEP program will be available during 2003, and we will consider this information when submitted, along with other information and comments we receive, before taking final action on the proposal.

Given the current data, however, we are confident that in applying the prospective RfC of 9 $mg/m^3$ to ambient annual average concentrations of 1 $mg/m^3$ or less, we may not reasonably anticipate MEK to cause chronic adverse human health effects. Neither may we reasonably anticipate adverse effects to occur

from short-term exposures.

E. Ecological Risk Characterization and Conclusions

Our review of the petition's ecological risk characterization supports the findings that MEK has limited persistence in water, soil, and air. We further agree that it has a low octanol/water coefficient, a low adsorption coefficient, and a low bioconcentration factor; therefore, given the available data, it is not anticipated to persist or accumulate in the environment.

A review of the general literature, including EPA databases, indicates that MEK has low environmental toxicity. For example, the daphnid 48-hour lethal concentrations for 50 percent of the testing sample (LC50) range from 2,200 to 5,091 ppm; the green algae 96-hour effective concentration for 50 percent of the population is 1,200 ppm; and the fish 96-hour LC50 ranges from 2,300 to 3,200 ppm. The fish chronic values range is 220 ppm, the daphnid chronic value is 521 ppm, and the algal chronic value is 45 ppm. These concentrations are significantly higher than what we would expect to see in the environment.

The petition included no data on the potential for ecological effects to occur as a result of exposures to media

other than water. There are no available data on avian exposure to MEK from the air pathway. There are also no available data on air exposure to plants from MEK. However, there is a database on laboratory mammals regarding air exposures to MEK from which we routinely extrapolate to draw conclusions regarding potential health effects to humans. From this database, we draw a similar conclusion regarding the potential for adverse health effects in mammals that may be exposed to ambient levels of MEK as we did for humans.

Based on our review of all pertinent data supplemented by additional environmental modeling, we have concluded that there are sufficiently adequate data on environmental effects of MEK to determine that ambient concentrations, bioaccumulation, or deposition of MEK may not reasonably be anticipated to cause adverse environmental effects.

F. Transformation Characterization

Methyl Ethyl Ketone is one of several volatile organic compounds (VOC) that transform into acetaldehyde and formaldehyde in the ambient air. Both acetaldehyde and formaldehyde are HAP and classified as probable human carcinogens. Based on a simplified analysis, the petitioner concluded that the contribution to ambient concentrations of

acetaldehyde and formaldehyde from MEK transformation is insignificant. This conclusion is largely based on the knowledge that MEK's half-life, about 9 days, is comparatively longer than its transformation products, acetaldehyde and formaldehyde, whose half-lives are about 14 hours and 3 hours, respectively. This implies that MEK's transformation products disappear much faster than they are formed. Our evaluation, summarized below, concurs with the petitioner's conclusion that atmospheric transformation of MEK emissions may not reasonably be anticipated to cause adverse effects to human health.

First, we assessed whether there would be elevated ambient concentrations near individual sources of MEK. Next, we estimated the ambient concentrations of these HAP resulting from transformation of MEK from multiple sources in urban areas. We then estimated the potential for any of these concentrations to cause adverse human health effects. Since the atmospheric chemistry for these pollutants is complex and not fully understood, we made conservative assumptions in the analysis in order to over- rather than under-estimate the concentrations of acetaldehyde and formaldehyde that could result from MEK transformation. Please refer to the docket for our complete analysis.

In the first step of the analysis, we applied tier 1 dispersion modeling (SCREEN3) to the worst-case facility presented in the petition, and assuming a conservative average wind speed of 3 miles per hour, we determined that the MEK plume from any given source will travel about 650 miles over MEK's 9-day half-life. Even at one tenth this duration (i.e., about 21 hours), still assuming a wind speed of 3 miles per hour, the plume will have traveled about 63 miles. In this plume, we estimated the unreacted MEK concentration after 21 hours to be approximately $1.6 \times 10^{-3}$ mg/m$^3$.

As it disperses, MEK transforms relatively slowly into acetaldehyde and formaldehyde and, in turn, these compounds decompose much more quickly into by-products, including carbon dioxide, carbon monoxide, and water. We estimated that about 7 percent of the MEK would have transformed into acetaldehyde and possibly formaldehyde after 21 hours. Accordingly, we estimated that the maximum concentrations of acetaldehyde and formaldehyde due to MEK transformation at this point (21 hours after being emitted) would be roughly $7 \times 10^{-5}$ mg/m$^3$ and $5 \times 10^{-5}$ mg/m$^3$, respectively. For acetaldehyde, that translates into a lifetime excess cancer risk of $1 \times 10^{-7}$. For formaldehyde, the lifetime excess cancer risk is $7 \times 10^{-7}$. Calculating noncancer hazard

quotients, we see that the HQ for acetaldehyde is 0.008. This means that the level of acetaldehyde to which people are exposed is 0.8 percent of the RfC. For formaldehyde, the HQ is 0.005, which means that the exposure level is 0.5 percent of the appropriate reference level, the Maximum Risk Level (MRL)[1]. Thus, since the cancer risks associated with the transformation products are below 1 in 1 million, and the noncancer exposures are less than 1 percent of the reference concentrations, we may not reasonably anticipate adverse health effects to occur from transformation of MEK into acetaldehyde and formaldehyde around MEK emissions sources. We note here that risk levels in the upwind part of the plume (i.e., the risks from the transformation products close to MEK emission sources) must be lower than what we estimated since the analysis did not account for degradation of acetaldehyde and formaldehyde. Further, we note that typical ambient levels of MEK are higher than they are in the plume at this point, indicating that the “plume,” as such, would no longer exist, having already merged indistinguishably with the ambient background. This turns our

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[1]There is no EPA RfC for formaldehyde. However, the Agency for Toxics Substances and Disease Registry has calculated a noncancer health effects level, called a MRL. The MRL for formaldehyde is 0.01 $mg/m^3$.

attention to the analysis of transformation products in the ambient background.

To evaluate the potential of acetaldehyde and formaldehyde to form from ambient concentrations of MEK significantly downwind of multiple emission sources, we looked at ambient monitoring data to determine the typical ambient level of MEK in urban environments. We then estimated the maximum concentrations of acetaldehyde and formaldehyde that could be transformed from this MEK, using conservative, steady-state assumptions. Based on available monitoring information, we determined that at the $95^{th}$ percentile, the ambient concentration of MEK is $4.3 \times 10^{-3}$ mg/$m^3$. Using an estimated degradation rate of 14 times greater than MEK for acetaldehyde, we estimated the ambient concentrations of acetaldehyde from transformed MEK to be $1.8 \times 10^{-4}$ mg/$m^3$. For formaldehyde, we estimated that it degrades at a rate of 72 times faster than MEK and, thus, calculated that the ambient concentration due to MEK transformation is $2 \times 10^{-5}$ mg/$m^3$. These very small concentrations do not represent significant health threats as they translate into lifetime excess cancer risks of $4 \times 10^{-7}$ for acetaldehyde and $3 \times 10^{-7}$ for formaldehyde.

We do not expect adverse noncancer health effects to occur

from the transformation of MEK. The HQ for acetaldehyde is 0.02 which corresponds to an exposure which is 2 percent of the RfC. For formaldehyde, the resulting HQ is $2x10^{-3}$ which represents an exposure of 0.2 percent of the MRL. Therefore, we may not reasonably anticipate adverse noncancer effects to occur due to exposures to these outdoor ambient concentrations of acetaldehyde or formaldehyde. Based on the analysis, we conclude that atmospheric transformation of MEK into acetaldehyde and formaldehyde may not reasonably be anticipated to cause significant human health risks.

G. Public Comments and EPA Responses

As part of the notice announcing receipt of a complete petition to delist MEK (64 FR 33453, June 23, 1999), we requested interested parties to provide us with data or comments. Copies of the public comments have been included in the docket for this action and have been considered in our review of the petition. Substantive comments are discussed below.

Comment. One commenter expressed concern about the overall appropriateness of the IRIS RfC as a decision criterion for determining human health effects. The commenter maintained that the IRIS RfC is itself uncertain and, therefore, the

petitioner's proposed revision is without merit. To support this position, data from a single long-term toxicity study which included MEK was cited. That study was published since the IRIS validation and reports adverse health effects as measured by decreased neural condition velocities for a set of workers (41 exposed, 63 controls) exposed over a period of 14 ± 7.5 years to levels of MEK ranging from 149 to 342 mg/m$^3$.

Response. The EPA's National Center for Environmental Assessment (NCEA) and National Health and Environmental Effects Research Laboratory (NHEERL) reviewed the referenced study as a part of our technical review. Their review demonstrated that the study has multiple and serious methodological shortcomings that greatly reduce its meaningfulness. Very few methodological details were presented in the study, making it virtually impossible for EPA reviewers to determine what had been done. It is not clear what factors were "matched" when the control groups were selected or how comparable the groups were on factors other than age. In addition, the study did not include important factors that are relevant to interpreting the results, including such factors as the type of work (e.g., office versus physical work); lifestyle factors (e.g., drinking, smoking, etc.); and height and weight of the subjects (important for

nerve conduction). Also, the study did not specify the experimental procedures that it applied, including whether the subjects were tested at the same location and time as the exposed workers, or whether the examiners were aware of the exposure status of the subjects at the time of testing. Importantly, the study did not address the control of temperature, a critical factor in nerve conduction studies, and the reported pattern of nerve conduction results is not entirely consistent with the reported peripheral neuropathy.

Of primary importance in EPA's review was the consideration of the extent to which the study's findings are supported by the existing scientific literature. In this regard, we conclude that the study cited in the comment is inconsistent with a large volume of high quality neurotoxicological scientific evidence. In fact, animal models of the reported condition are excellent predictors of human neuropathy. MEK has been well tested for the reported condition and is convincingly negative.

Comment. The EPA received a comment expressing concern over MEK's role in potentiating the effect of other substances. The comment stated that given the "ubiquitous" ambient concentration of certain pollutants and general lack of

understanding of the mechanisms of potentiation, it would be inappropriate for the Agency to allow an increase in ambient concentrations of MEK.

Response. As described in this preamble, MEK has been shown to potentiate neurotoxicity of other solvents in experiments with laboratory animals when both are present in high concentrations. The lower limits of MEK exposure that may result in potentiation with other solvents have not been well established in animals, and the potential of MEK in this regard remains a concern, although a minor one.

H. Other Issues

Since the receipt of the petition, MEK has been measured in the blood of the general population as reported from the National Health and Nutrition Examination Survey (NHANES) database. The NHANES database reports median blood levels of 5.4 parts per billion (ppb) and levels at the $95^{th}$ percentile of 16.9 ppb. The EPA estimates that it would take continuous exposures at ambient concentrations near 1 $mg/m^3$ of MEK to result in the reported median blood level.

However, based on the available information, EPA believes it is reasonable to expect that the reported blood levels did not result from an air exposure to MEK at the prospective RfC.

Primarily, this is because concentrations of MEK found in the immediate vicinity of large MEK emissions sources are below the RfC, and as previously stated in today's action, typical ambient background concentrations of MEK are several orders of magnitude lower than the prospective RfC.

In addition, although MEK has been shown in animal studies to be readily absorbed, it is also rapidly metabolized, mostly in the liver. The excretion half-life of MEK is quite short, on the order of minutes to hours (Liira et al., 1988), and is nearly quantitatively complete in both animals and in humans. The data indicate that internal doses following experimental air exposures to MEK consist mostly of metabolites that are cleared quickly. Therefore, tissue and blood levels of MEK would become minimal shortly after termination of experimental air exposures due to kinetics and solubility of MEK. Likewise, for those persons exposed to relatively high concentrations of MEK, blood levels would fall relatively quickly to pre-exposure levels following the termination of exposure.

Consequently, it is the judgment of scientists from both the Centers for Disease Control (CDC), who compiled the NHANES database, and EPA that the data are not representative of atmospheric exposure of national proportions. These authors

also state that blood levels of both MEK and acetone are highly variable as a result of their physiologic metabolism and do not reflect environmental exposures very well (Churchill et al., 2001). Thus, it is more likely that the reported MEK in human blood is a by-product of normal human metabolism.

Another issue we addressed in today's action is that of MEK as an ozone precursor. The EPA recognizes that MEK is an ozone precursor, but after considering this issue, we determined that it is inappropriate to include a substance on the HAP list under CAA section 112(b) due entirely to its tendency to form ozone. Section 112(b) provides that no air pollutant which is listed under CAA section 108(a), such as ozone, may be added to the HAP list. It further provides that a pollutant that is a precursor to a pollutant listed under section 108(a), such as MEK, may not be included on the HAP list unless it independently meets the HAP list criteria. As explained in today's action, we believe that the petitioner has demonstrated that MEK does not independently meet the criteria for listing as a HAP under section 112 of the CAA.

The Agency has previously determined that MEK could not be removed from the list of pollutants under section 313 of the Emergency Planning and Community Right-To-Know Act of 1986

(EPCRA) (63 FR 15195). However, the EPCRA list serves a very different purpose than the list of HAP under section 112(b) of the CAA. Specifically, the EPCRA–-which is intended to provide information regarding the emissions of air pollutants generally–-deals collectively with HAP, VOC, and other air and water pollutants under section 313 by providing for the listing of any pollutant that may reasonably be anticipated to cause adverse effects to human health or the environment. The CAA, on the other hand, establishes requirements for reducing the emissions of air pollutants and deals separately with HAP (which are to be listed and regulated under section 112) and criteria air pollutants (which are to be listed under section 108 and regulated under various other sections of the CAA). The EPA is required to regulate precursors to criteria air pollutants, such as VOC, for their contributions to ambient levels of criteria pollutants under statutory provisions that do not apply to HAP. This dual structure would lose its significance if EPA were to include substances on the HAP list solely as a result of their contribution to concentrations of criteria air pollutants.

The decision to grant the petition and issue a proposed rule to delist MEK removes MEK from regulatory consideration under section 112(d) of the CAA. Section 112 requires the

development of maximum achievable control technology (MACT) standards to reduce routine emissions of listed toxic air pollutants. The proposed rule does not affect MEK's status under the CAA as a VOC, and EPA will continue to regulate it as such. In ozone nonattainment areas, sources of MEK emissions must continue to meet applicable standards identified in State implementation plans (SIP).

In addition, the proposed rule does not impact any MEK reporting requirements under the TRI (EPCRA, section 313). Recognizing that MEK is one of the largest sources listed in the TRI, the Agency will continue to track emissions of MEK. Further, under the CAA, the Agency has the option to add MEK back onto the HAP list and will do so should a need arise.

I. Discussion and Conclusion

Uncertainty is an inherent part of risk assessment. It arises because risk assessment is a complex process, requiring the integration of multiple factors. In the analysis, uncertainty arises for the following reasons. The IRIS dataset used to derive the human health effects decision criterion is imperfect and leads to uncertainty in the RfC. This uncertainty is primarily due to the lack of long-term MEK toxicity data and is compensated for in the application of an uncertainty factor

of 100 for the prospective RfC. In addition, animal cancer bioassays with MEK by either the oral or inhalation route are lacking from the database, and there is scientific uncertainty in MEK's ability to potentiate the action of other neurotoxins. We also recognize that there is uncertainty in the computer models used to predict the fate and transport of MEK in the environment. These models are simplifications of reality and some variables are excluded.

For decisions which are based largely on risk assessments, some degree of uncertainty is acceptable. Such is the case for this delisting decision. We do not interpret CAA section 112(b)(3)(C) to require absolute certainty that a pollutant will not cause adverse effects on human health or the environment before it may be deleted from the list. The use of the terms "adequate" and "reasonably" indicate that the Agency must weigh the potential uncertainties and their likely significance. To this end, the assessment applies conservative assumptions to bias potential error toward protecting human and ecological health. Thus, EPA is confident that even when we consider the uncertainties in the petition's initial assessment and in the additional analyses, the results are more likely to over-estimate rather than under-estimate true exposures and risks.

Based on our evaluation of the petition and the subsequent analyses, we judge that the potential for adverse human health and environmental effects to occur from projected exposures is sufficiently low to provide reasonable assurance that such adverse effects will not occur. For example, the petitioner appropriately applied EPA's model guidelines and EPA's tiered dispersion modeling approach which we designed to be conservative. Also, EPA suggested that the petitioner conduct an additional, more site-specific analysis to verify the conservatism of the original analysis. The results of that analysis increased our confidence that the petition over- rather than under-estimates exposure. In addition, the petition did not apply a formal exposure assessment to the predicted ambient air concentrations. Instead, the petition used the air concentrations alone as a surrogate for exposure. Based upon the likely proximity of inhabitable areas and knowledge of human activity patterns, we believe that actual exposures will be far less than predicted exposures that were derived from the dispersion analysis. Further, when modeling clusters of MEK sources, the petition showed that concentrations resulting from that scenario are not likely to adversely affect health. Finally, available data from monitors suggest that ambient

concentrations of MEK in urban areas are over two orders of magnitude lower than the modeled maximum concentrations.

As described above, EPA's proposed decision to delist MEK is based on the results of a risk assessment demonstrating that emissions of MEK may not reasonably be anticipated to result in adverse human health or environmental effects. In addition to the analyses presented and the uncertainties inherent in risk assessment, we have considered other information related to MEK in making this decision, namely the transformation of MEK into acetaldehyde and formaldehyde and recently discovered levels of MEK in human blood. The MEK decomposes in the ambient air into two probable human carcinogens (acetaldehyde and formaldehyde). However, given that the actual contribution of MEK to ambient concentrations of these two pollutants is very small, and that they decompose rapidly, we do not anticipate that MEK transformation into these two pollutants will be significant enough to have an adverse impact on human health. We do not expect that ambient concentrations of MEK contribute significantly to the blood level burden due to the small ambient concentrations of MEK in ambient air.

We also considered the fact that MEK is one of the top compounds by volume reported in the TRI. Under this proposal,

it would no longer be regulated as a HAP, but it will continue to be reported in the TRI and regulated under EPA's criteria pollutant (ozone) program.

As discussed previously, we will consider the RfC that results from the IRIS review and information combined in industry's submission under tier 1 of the VCCEP before taking final action on the proposal. We also welcome additional data or information that can further clarify these and other issues related to MEK. We will evaluate all substantive information received during the comment period prior to taking any final action on the proposed rule.

## V. References

References cited in the preamble can be viewed in the docket for this proposed rule.

## VI. Statutory and Executive Order Reviews

### A. Executive Order 12866: Regulatory Planning and Review

Under Executive Order 12866 (58 FR 51735, October 4, 1993), EPA must determine whether the regulatory action is "significant" and therefore subject to Office of Management and Budget (OMB) review and the requirements of the Executive Order. The Executive Order defines "significant regulatory action" as one that is likely to result in a rule that may:

(1) Have an annual effect on the economy of $100 million or more or adverse affect in a material way the economy, a sector to the economy, productivity, competition, jobs, the environment, public health or safety, or state, local or tribal governments or communities;

(2) create a serious inconsistency or otherwise interfere with an action taken or planned by another agency;

(3) materially alter the budgetary impact of entitlements, grants, user fees, or loan programs, or the rights and obligation of recipients thereof; or

(4) raise novel legal or policy issues arising out of legal mandates, the President's priorities, or the principles set forth in the Executive Order.

Pursuant to the terms of Executive Order 12866, it has been determined that the proposed action does not constitute a "significant regulatory action" and is, therefore, not subject to OMB review.

B. Paperwork Reduction Act

This action does not impose an information collection burden under the provisions of the Paperwork Reduction Act, 44 U.S.C. 3501 et seq. The proposed action will remove MEK from the CAA section 112 (b)(1) HAP list and, therefore, eliminate

the need for information collection under the CAA. Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information. An Agency may not conduct or sponsor, and a person is not required to respond to a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations are listed in 40 CFR part 9 and 48 CFR chapter 15.

C. Regulatory Flexibility Act (RFA)

The RFA generally requires an agency to prepare a regulatory flexibility analysis of any rule subject to notice and comment rulemaking requirements under the Administrative

Procedure Act or any other statute unless the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities. Small entities include small business, small organizations, and small governmental jurisdictions. For the purposes of assessing the impacts of today's proposed rule on small entities, small entity is defined as: (1) a small business that meets the definitions for small business based on the Small Business Association (SBA) size standards which, for this proposed action, can include manufacturing (NAICS 3999-03) and air transportation (NAICS 4522-98 and 4512-98) operations that employ less 1,000 people and engineering services (NAICS 8711-98) operations that earn less than $20 million annually; (2) a small governmental jurisdiction that is a government of a city, county, town, school district or special district with a population of less than 50,000; and (3) a small organization that is any not-for-profit enterprise which is independently owned and operated and is not dominant in its field.

After considering the economic impact of today's proposed rule on small entities, I certify that this proposed action will not have a significant economic impact on a substantial number of small entities. In determining whether a rule has

significant economic impact on a substantial number of small entities, the impact of concern is any significant adverse economic impact on small entities, since the primary purpose of the regulatory flexibility analysis is to identify and address regulatory alternatives “which minimize any significant economic impact of the proposed rule on small entities.” (5 U.S.C. 603 and 604). Thus, an agency may certify that a rule will not have a significant economic impact on a substantial number of small entities if the rule relieves regulatory burden, or otherwise has a positive economic effect on all of the small entities subject to the rule. The proposed rule will eliminate the burden of additional controls necessary to reduce MEK emissions and the associated operating, monitoring and reporting requirements. We have, therefore, concluded that today’s proposed rule will relieve regulatory burden for all small entities. We continue to be interested in the potential impacts of the proposed rule on small entities and welcome comments on issues related to such impacts.

D. Unfunded Mandates Reform Act

Title II of the Unfunded Mandates Reform Act of 1995 (UMRA), Public Law 1044, establishes requirements for Federal agencies to assess the effects of their regulatory actions on

State, local, and tribal governments and the private sector. Under section 202 of the UMRA, EPA generally must prepare a written statement, including a cost-benefit analysis, for proposed and final rules with “Federal mandates” that may result in expenditures to State, local, and tribal governments, in the aggregate, or to the private sector, of $100 million or more in any 1 year. Before promulgating an EPA rule for which a written statement is needed, section 205 of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective or least burdensome alternative that achieves the objectives of the rule. The provisions of section 205 do not apply when they are inconsistent with applicable law. Moreover, section 205 allows EPA to adopt an alternative other than the least costly, most cost-effective or least burdensome alternative if the Administrator publishes with the final rule an explanation why that alternative was not adopted. Before EPA establishes any regulatory requirements that may significantly or uniquely affect small governments, including tribal governments, it must have developed under section 203 of the UMRA a small government agency plan. The plan must provide for notifying potentially affected small governments, enabling officials of affected small

governments to have meaningful and timely input in the development of EPA regulatory proposals with significant Federal intergovernmental mandates, and informing, educating, and advising small governments on compliance with the regulatory requirements.

Today's proposed rule contains no Federal mandates for State, local, or tribal governments or the private sector. The proposed rule imposes no enforceable duty on any State, local or tribal governments or the private sector. In any event, EPA has determined that the proposed rule does not contain a Federal mandate that may result in expenditures of $100 million or more for State, local, and tribal governments, in the aggregate, or the private sector in any 1 year. Because the proposed rule removes a compound previously labeled in the CAA as a HAP, it actually reduces the burden established under the CAA. Thus, today's proposed rule is not subject to the requirements of sections 202 and 205 of the UMRA.

E. Executive Order 13132, Federalism

Executive Order 13132 (64 FR 43255, August 10, 1999) requires EPA to develop an accountable process to ensure "meaningful and timely input by State and local officials in the development of regulatory policies that have federalism

implications." "Policies that have federalism implications" is defined in the Executive Order to include regulations that have "substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government."

Under Executive Order 13132, EPA may not issue a regulation that has federalism implications, that imposes substantial direct compliance costs, and that is not required by statute, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by State and local governments, or EPA consults with State and local officials early in the process of developing the proposed regulation. The EPA also may not issue a regulation that has federalism implications and that preempts State law unless the Agency consults with State and local officials early in the process of developing the proposed regulation.

Today's proposed rule removes the substance MEK from the list of HAP contained under section 112(b)(1) of the CAA. It does not impose any additional requirements on the States and does not affect the balance of power between the States and the Federal government. Thus, the requirements of section 6 of the

Executive Order do not apply to the proposed rule.

F. Executive Order 13175, Consultation and Coordination with Indian Tribal Governments

Executive Order 13175, entitled “Consultation and Coordination with Indian Tribal Governments” (65 FR 67249, November 9, 2000), requires EPA to develop an accountable process to ensure “meaningful and timely input by tribal officials in the development of regulatory policies that have tribal implications.” The proposed rule does not have tribal implications, as specified in Executive Order 13175.

A review of the available emission inventory does not indicate tribal MEK emissions sources subject to control under the CAA, therefore, the proposed rule is not anticipated to have tribal implications. In addition, the proposed action will eliminate control requirements for MEK and, therefore, reduces control costs and reporting requirements for any tribal entity operating a MEK source subject to control under the CAA which we might have missed. Thus, Executive Order 13175 does not apply to the proposed rule.

G. Executive Order 13045, Protection of Children from Environmental Health Risks and Safety Risks

Executive Order 13045 (62 FR 19885, April 23, 1997)

applies to any rule that: (1) is determined to be "economically significant" as defined under Executive Order 12866, and (2) concerns an environmental health or safety risk that EPA has reason to believe may have a disproportionate effect on children. If the regulatory action meets both criteria, the Agency must evaluate the environmental health or safety effects of the planned rule on children, and explain why the planned regulation is preferable to other potentially effective and reasonably feasible alternatives considered by the Agency.

The EPA interprets Executive Order 13045 as applying only to those regulatory actions that are based on health or safety risks, such that the analysis required under section 5-501 of the Executive Order has the potential to influence the regulation. The proposed rule is not subject to Executive Order 13045 because it is not economically significant as defined in Executive Order 12866, and because the Agency does not have reason to believe the environmental health or safety risks addressed by this action present a disproportionate risk to children. This determination is based on the fact that the RfC is determined to be protective of sensitive sub-populations, including children. Also, the single study cited during public comment to indicate a potential effect on children has been

reviewed during this petition process and found to be limited in design and execution. Consequently, we determined that the study was of insufficient quality to provide information regarding health risks (leukemia) of MEK to children. However, as we state above, we anticipate industry's submission to the first tier of the VCCEP program will be available during 2003, and we will consider this information when submitted. In addition, the public is invited to submit or identify peer-reviewed studies and data, of which the Agency may not be aware, that assessed results of early life exposure to MEK.

H. Executive Order 13211, Actions Concerning Regulations that Significantly Affect Energy Supply, Distribution, or Use

Executive Order 13211, "Actions Concerning Regulations that Significantly Affect Energy Supply, Distribution, or Use" (66 FR 28355, May 22, 2001), requires EPA to prepare and submit a Statement of Energy Effects to the Administrator of the Office of Information and Regulatory Affairs, Office of Management and Budget, for certain actions identified as "significant energy actions." The proposed rule is not a "significant energy action" because it is not likely to have a significant adverse effect on the supply, distribution, or use of energy.

I. National Technology Transfer and Advancement Act

Section 112(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law No. 104-113, section 12(d) 915 U.S.C. 272 note), directs all Federal agencies to use voluntary consensus standards instead of government-unique standards in their regulatory activities unless to do so would be inconsistent with applicable law or otherwise impractical. Voluntary consensus standards are technical standards (e.g., material specifications, test method, sampling and analytical procedures, business practices, etc.) that are developed or adopted by one or more voluntary consensus standards bodies. Examples of organizations generally regarded as voluntary consensus standards bodies include the American society for Testing and Materials (ASTM), the National Fire Protection Association (NFPA), and the Society of Automotive Engineers (SAE). The NTTAA requires Federal agencies like EPA to provide Congress, through OMB, with explanations when an agency decides not to use available and applicable voluntary consensus standards. The proposed rule does not involve technical standards. Therefore, EPA is not considering the use of any voluntary consensus standards.

List of Subjects in 40 CFR Part 63

Environmental protection, Air pollution control, Hazardous substances, Reporting and recordkeeping requirements.

_____
Date

_____
Christine Todd Whitman
Administrator

For the reasons set out in the preamble, part 63, title 40, chapter I of the Code of Federal Regulations is proposed to be amended as follows:

**PART 63– NATIONAL EMISSION STANDARDS FOR HAZARDOUS AIR POLLUTANTS FOR SOURCE CATEGORIES**

1. The authority citation for part 63 continues to read as follows:

Authority: 42 U.S.C. 7401, et seq.

**Subpart C-[AMENDED]**

2. Subpart C is amended by adding §63.61 and reserving §§63.62 through 63.69 to read as follows:

**§63.61 Deletion of methyl ethyl ketone from the list of hazardous air pollutants.**

The substance methyl ethyl ketone (MEK, 2-Butanone) (CAS Number 105602) is deleted form the list of hazardous air pollutants established by 42 U.S.C. 7412(b)(1).

§§63.62-63.69 **[Reserved]**